Left ventricular pressure–area relations as assessed by transoesophageal echocardiographic automated border detection: comparison with conductance catheter technique in cardiac surgical patients

D. Schmidlin[1]*, S. Aschkenasy[1], P. R. Vogt[2], J. Schmidli[2], R. Jenni[3] and E. R. Schmid[1]

[1]*Institute of Anaesthesiology,* [2]*Clinic for Cardiovascular Surgery and* [3]*Department of Internal Medicine, University Hospital, CH-8091 Zurich, Switzerland*

*Corresponding author

The aim of this study was to validate measurements of intraoperative left ventricular (LV) area by transoesophageal echocardiography against simultaneous measurements of LV volume by conductance catheter (CC) in cardiac surgical patients with normal systolic LV function. Echo area was compared with CC volume during steady state and during acute changes of pre- and afterload by partial clamping of the inferior vena cava and the ascending aorta in eight patients scheduled for coronary artery bypass grafting. At steady state, Bland–Altman analysis of 32 recordings revealed a bias (SD) of 0.6% (2.5%) between echo area and CC volume, related to the initial values of end-diastolic area (100% area) and volume (100% volume), respectively. During loading interventions, bias between the two methods, as assessed by 112 measurement sequences, was 0.5% (3.7%) during aortic occlusion and –3.9% (4.4%) during cava occlusion at end-systole ($P < 0.0001$); at end-diastole, this bias was 1.3% (4%) during aortic occlusion and 0.2% (5.7%) during cava occlusion ($P < 0.0001$). Intraoperative area measurements with transoesophageal echocardiography in cardiac surgical patients with normal systolic LV function show good correlation with CC volume measurements under steady-state conditions. During acute unloading by vena cava occlusion, the resulting small end-systolic echo area measurements differ significantly more from CC volume measurements than during acute increase in afterload by aortic occlusion.

Left ventricular (LV) pressure–volume loops are a useful measure of LV performance in a relatively load-independent manner.[1–4] In one animal[5] and two human[6 7] studies, loops acquired with an LV conductance and an LV pressure catheter were very similar under various conditions to those acquired with echocardiographic automated border detection and LV pressure. However, most of these authors used an echocardiographic border detection system manufactured by a single company; intraoperative validation of echocardiographic automated border detection with the conductance catheter (CC) method in humans has been reported by one group, so far.[6]

The aim of this study was to validate in humans intraoperative area measurements obtained by another type of automated echocardiographic system, using digital echo quantification (DEQ) against simultaneous volume measurements, obtained by an LV CC. Comparison of the two methods during cardiac surgery was undertaken to investigate the strengths and limitations of the intraoperative use of the DEQ method in patients with normal systolic LV function. We hypothesized that, during steady state and acute changes in loading, area changes measured with transoesophageal echocardiography at the mid-papillary short axis level would reflect accurately changes in CC volume.

Materials and methods

Patient characteristics

Following ethics committee approval and written informed consent, measurements were performed before and after

cardiopulmonary bypass (CPB) in nine patients with stable coronary artery disease and normal LV ejection fraction undergoing coronary artery bypass grafting. None had akinetic or dyskinetic LV myocardial segments, two patients had discrete inferior hypokinesia and all subjects were in sinus rhythm. In one patient, echo quality in the short-axis view was not sufficient, so he was excluded from the study. The remaining eight patients (one female) had a mean age of 65 yr (95% confidence interval (CI) 61–69 yr) and an LV ejection fraction of 68% (95% CI 62–74%). Preoperative medication consisted of beta-blockers in seven patients, nitrates in four and calcium channel blockers in two; none of the patients was treated with ACE inhibitors.

Anaesthesia and operative techniques

Preoperative medication was continued until the morning of surgery. Patients were premedicated with oral flunitrazepam 0.02–0.03 mg kg^{-1}. In the operating room, a peripheral venous cannula and a fluid-filled femoral artery catheter (Seldicath, 1.3 mm; Plastimed, St-Leu-La-Forêt, France) were inserted under local anaesthesia. The arterial catheter was connected to a single-use transducer (Deltran II; Utah Medical Inc., Midvale, UT, USA). Continuous ECG (leads II and V5), arterial pressure and transcutaneous oxygen saturation monitoring were installed and connected to the monitoring system (Solar 8000; Marquette-Hellige GmbH, Freiburg, Germany). A continuous three-lead ECG was connected to the echo machine for DEQ signal-processing. Anaesthesia was induced with flunitrazepam (total dose 0.01–0.05 mg kg^{-1}) and fentanyl (total dose 25–55 µg kg^{-1}). Pancuronium 0.1 mg kg^{-1} was given to facilitate tracheal intubation, and patients' lungs were mechanically ventilated with a Servo 900C ventilator (Siemens Elema AB, Upplands Väsby, Sweden). Anaesthesia was maintained with propofol by continuous infusion (1.8–4.5 mg kg^{-1} h^{-1}). Central venous and pulmonary artery catheters (Baxter Intellicath 7.5 F; Baxter Healthcare Corp., Irvine, CA, USA) were placed via the right internal jugular vein, and the transoesophageal echo probe (see below) was inserted.

After sternotomy and pericardiotomy, epicardial pacemaker wires were fixed on the right atrium. After systemic heparinization, the aorta was cannulated, the CC was inserted via the right upper pulmonary vein and measurements were performed before right atrial cannulation.

Drug therapy for weaning from CPB consisted of nitroglycerine 0.6 (95% CI 0.26–0.94) µg kg^{-1} min^{-1} (six patients) or nifedipine 0.046 and 0.064 µg kg^{-1} min^{-1} (in two cases). In one case, norepinephrine 0.037 µg kg^{-1} min^{-1} was given for low systemic vascular resistance; no other catecholamine therapy was necessary in the study group. Propofol 3.3 (95% CI 2.6–4.1) mg kg^{-1} h^{-1} was used to maintain hypnosis during rewarming, chest closure and transfer to the intensive care unit.

Echocardiographic DEQ

Transoesophageal echocardiography with DEQ was done with an Omniplane 5 MHz echo probe and a Vingmed CFM 800 system (Vingmed Sound, Horten, Norway). We reduced overall gain, adjusted time gain, lateral gain compensations and compress levels and narrowed the image sector for optimal DEQ conditions and the highest possible frame rate (30–50 frames s^{-1}).[14] The DEQ uses high-quality two-dimensional (2D) images in a transgastric mid-papillary view; raw backscatter data are stored in memory and are subjected to a dynamic processing algorithm that applies statistically based edge-detection enhancement. A contour identifying the interface between blood and myocardium is superimposed on the real-time echocardiographic image and used for automated area calculation; it is displayed as a real-time trace in a manually tracked region of interest. The trace is exported as an analogue signal to an analogue–digital (A/D) converter and recorded with a sample rate of 1000 Hz on a workstation.

Conductance catheter

A 7 French, 12 electrode LV CC (Cordis Europa NV, LJ Roden, The Netherlands) with an integrated tip manometer (Sentron BV, AC Roden, The Netherlands) was introduced via the right upper pulmonary vein through the mitral valve with the help of an LV vent introducer and fixed with a purse-string suture. The catheter was advanced until four correct volume tracings were displayed on the screen of the CC monitoring system; its position within the left ventricle was controlled for centred radial position by transoesophageal echocardiography in a longitudinal view of the left ventricle. Tip manometer catheters were immersed in saline, then calibrated relative to atmospheric pressure; specific blood resistivity (ρ), which is defined as 1/blood conductance (σ), was measured before and after CPB with the help of a blood sample and the measuring cuvette supplied with the CC analysis system (Leycom Sigma 5 DF; CardioDynamics BV, Zoetermeer, The Netherlands). Injections of 12.5% saline into the distal port of the pulmonary artery catheter for determination of parallel conductance[2 3] were performed before and after CPB, immediately after determination of cardiac output with thermodilution (three to five injections of iced saline).

Data acquisition and registration

The ECG and femoral arterial pressure signals were amplified by the Solar 8000 monitoring system with a direct output to the A/D system. All analogue signals of interest (DEQ area, five CC volumes, micromanometer LV pressure, femoral arterial pressure and ECG) were digitized with a sample rate of 1000 Hz for display and stored with the help of an A/D card and customized software under the environment of Superscope II (MacAdios; GW Instruments, Somerville, MA, USA) on an Apple Macintosh computer.

Compensation of the time used by the echocardiography system for calculation and display of DEQ was achieved by time-advancing the DEQ area curve until the first three to five beats of one 15 beat run were congruent to the CC volume signal. Compensation of the general, system-related, and the individual physiological time delay of a fluid-filled catheter against a rapid response micromanometer was performed by advancing the femoral arterial pressure curve until its ejection phases exactly matched the ejection phases of the LV pressure curve of the micromanometer.

To reduce electrical noise, all signals were filtered with a low-pass HAM filter with a 50 Hz frequency cut-off, signals >50 Hz being attenuated and those ≤50 Hz left unchanged.

Protocol

Simultaneous CC volume, DEQ area, LV and femoral arterial pressure signals were obtained during apnoea at end-expiration with zero positive airway pressure for 30 s. After a baseline measurement, one or two runs with injection of hypertonic saline into the distal port of the pulmonary artery catheter were then recorded. Acute alterations in preload were induced by partially clamping the inferior vena cava and afterload was increased by a partial aortic cross-clamping manoeuvre ('aortic occlusion'). Before CPB and after weaning from CPB and haemodynamic stabilization, we obtained duplicate measurements 5 min apart (i) during caval (Fig. 1A) and aortic occlusion (Fig. 1B) while the patient was in sinus rhythm, and (ii) with atrial pacing at a heart rate of 90 bpm. The registered LV pressure–volume and LV pressure–area loops were analysed off-line.

Data analysis

For steady-state analysis of DEQ area and CC volume, four runs per patient with optimal DEQ signal quality were chosen. Within these runs, three beats at the beginning or the end of the corresponding manoeuvre were analysed at 5 ms intervals, i.e. at a sampling rate of 200 Hz. For presentation of area data in cm^2, the stored analogue DEQ data were recalibrated using the end-diastolic and end-systolic values stored in the echocardiography system. Raw analogue output of the CC volume was calibrated for effective volume using the correction factor α with the help of the thermodilution-derived stroke volume as described previously.[4]

DEQ area and CC volume were normalized based on the first end-diastolic DEQ area (100% area) and CC volume (100% volume) values (Figures 3A and 4); values measured subsequently were expressed as a percentage of the initial end-diastolic values.

From individual runs during acute loading changes, 15 beats were selected: three to five beats before LV pressure changes and 10–12 during loading alteration.

For visual analysis, the DEQ signal (in mV) was calibrated for amplitude, baseline offset and time delay;
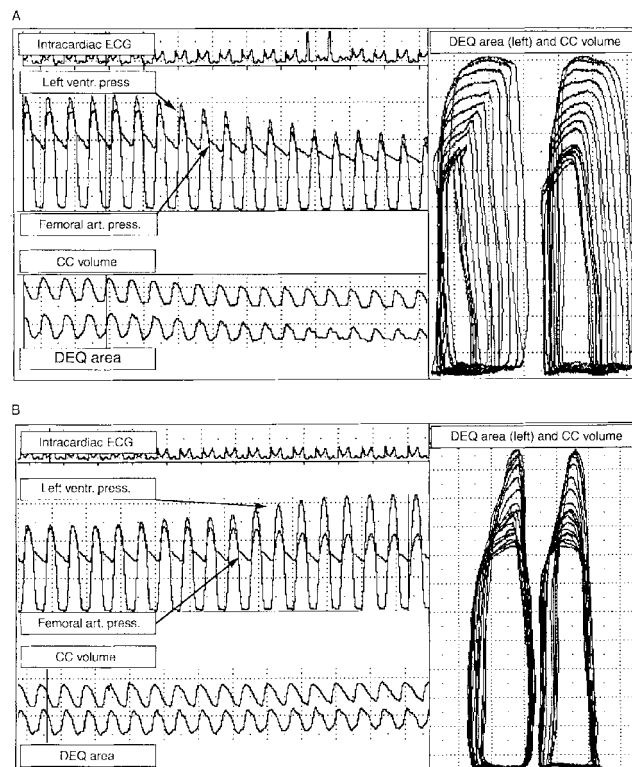

Fig 1 Manoeuvres for acute loading change: partial clamping of (A) inferior vena cava and (B) ascending aorta. ECG=electrocardiogram; left ventr press=left ventricular pressure from the conductance catheter tip micromanometer; CC volume=conductance catheter volume; DEQ area=digital echo quantification.

the femoral arterial pressure curve was adjusted in time as described above. The LV pressure–area and LV pressure–volume loops were displayed for the selected beats. End-systolic and end-diastolic points of each loop were analysed. End-systole was measured at aortic valve closure, as determined by the femoral arterial pressure curve (Fig. 1), and at end-diastole at the end of the diastolic pressure plateau.

End-systolic and end-diastolic slopes of LV pressure plotted against DEQ area and CC volume, respectively, were calculated by linear regression analysis. All values of interest were exported to a data sheet for statistical analysis.

Statistics

Raw data are expressed as median and 95% CI; Bland–Altman analyses in the text are reported as mean (SD). Bland–Altman analyses and one linear regression analysis were used to present correlation of DEQ area and CC volume data. The significance of differences between measurements was determined using the Wilcoxon ranked sign test for paired comparisons and the Mann–Whitney U-test for unpaired comparisons. P values <0.05 were considered significant. Calculations were made with Statview 4.5 (Abacus Concepts Inc, Berkeley, CA, USA).
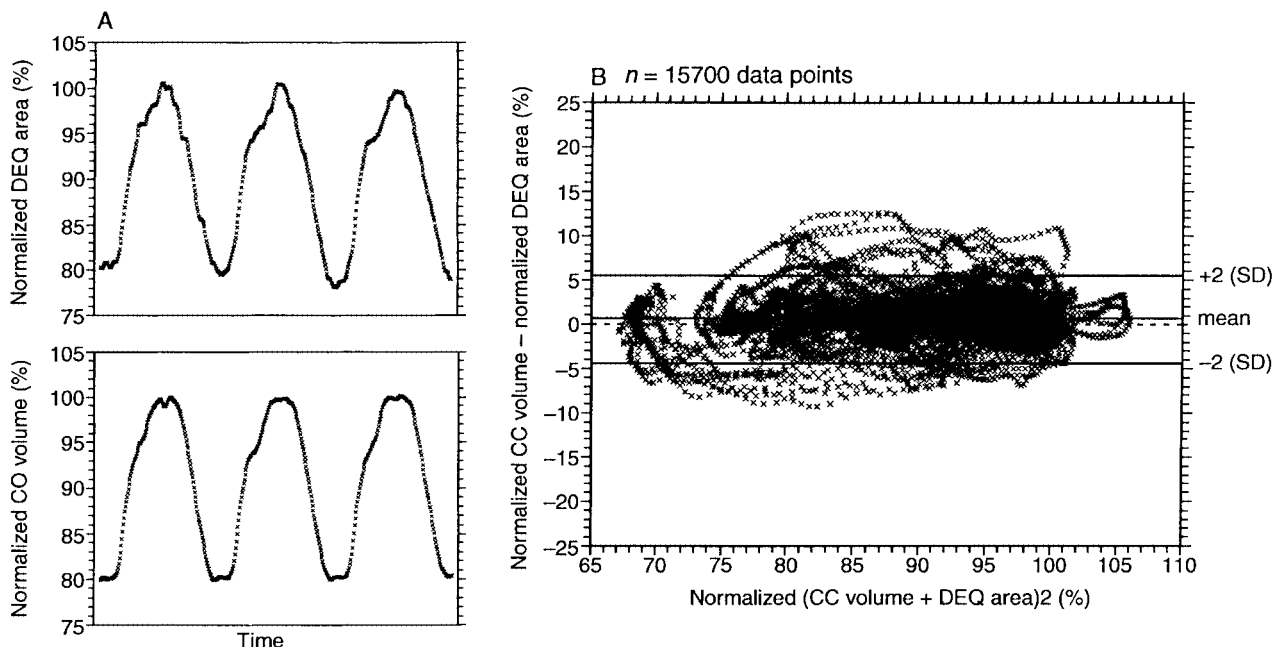
Fig 2 Simultaneous measurement of digital echo quantification (DEQ) area and conduance catheter (CC) volume at steady-state conditions. Three consecutive beats per patient are shown. Normalized DEQ area (%) and normalized CC volume (ml) are compared. Measurements were taken every 5 ms. (A) Example of a steady-state recording of normalized DEQ area (above) and normalized CC volume (below) during three cardiac cycles, adjusted for calculation time of DEQ area. (B) Bland–Altman analysis of normalized DEQ area and normalized CC volume. n=15 700 data pairs, 32 measurements (four per patient). For numerical values, see text.

Results

General

A total of 174 measurements was performed. Forty-seven (27%) were not considered for analysis, 22 (12.6%) for technical reasons (improper partial clamping or declamping manoeuvre) and 25 (14.4%) because of insufficient transoesophageal echocardiograph image quality. Fifteen good-quality runs were randomly chosen not to be analysed for statistical reasons (not more than 16 runs per patient).

A total of 112 measurement runs of 15 beats each (median 14, 95% CI 12–14 runs per patient) was analysed during manoeuvres for acute loading changes (Fig. 1). For visual comparison with the CC volume data, amplification of the area signal was 9.5 (95% CI 8.4–10.6) times, baseline offset 7.2 (95% CI 4.8–9.6) mV and the area signal time offset compared with the CC volume signal was –194 (95% CI –202 to –168) ms.

Steady-state correlation of DES area and CC volume measurements

During steady state, curves of area changes as registered by DEQ showed a close agreement with curves obtained by the CC technique (Fig. 2): Figure 2A shows an example of parallel registration and Fig. 2B a Bland–Altman analysis of 32 steady-state recordings at 5 ms intervals; the mean (SD)

difference between normalized DEQ area and normalized CC volume was 0.6 (2.5)%.

The difference between normalized end-systolic DEQ area and CC volume of these steady-state recordings was 1.0 (1.7)%; the end-diastolic difference was 0.3 (1.3)%.
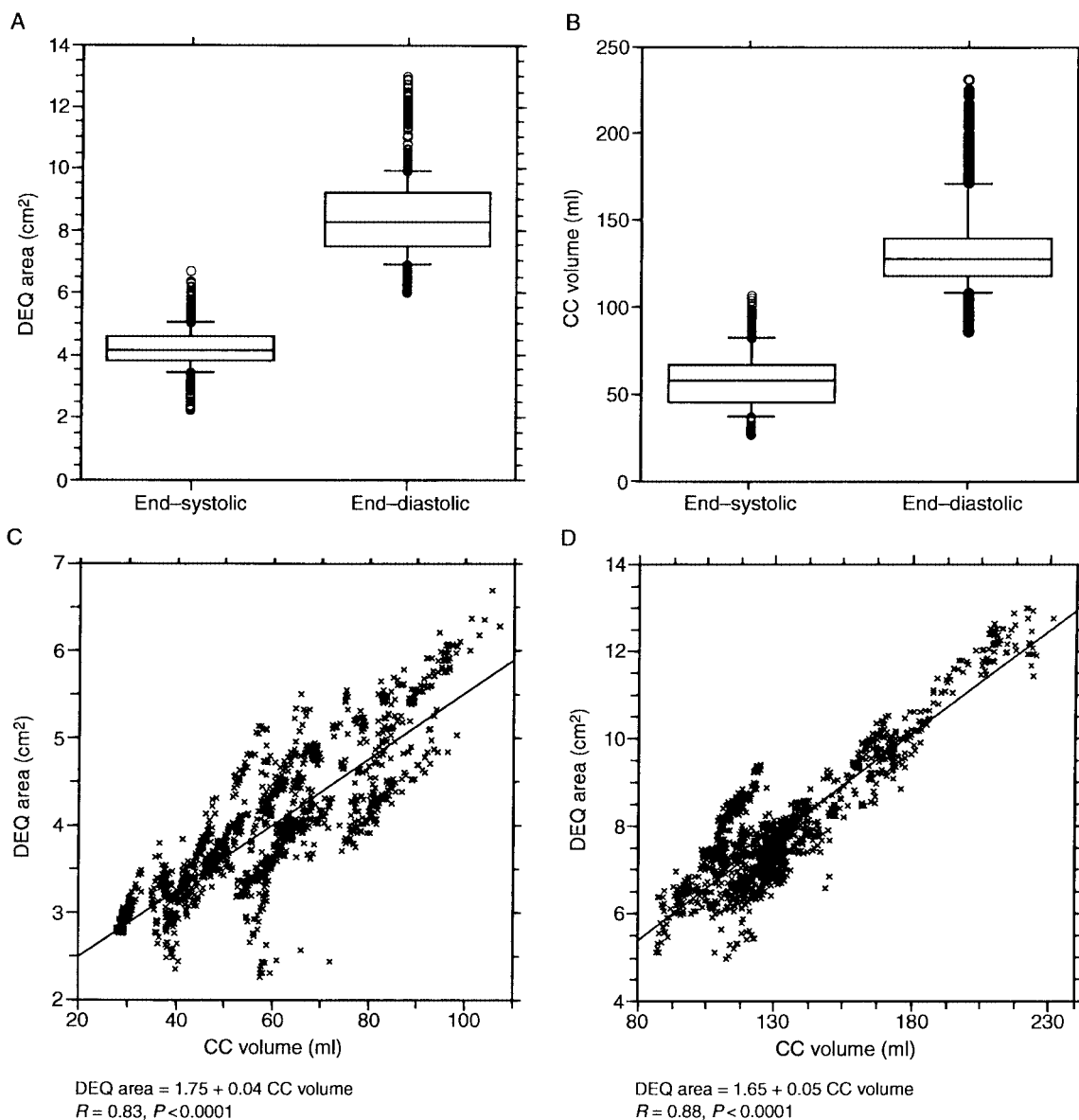
Correlation during interventions

DEQ area versus CC volume

Calibrated absolute DEQ area and CC volume values are presented as box plots (Fig. 3A,B) and as linear regression analyses (Fig. 3C,D). Numeric values are shown in Table 1.

DEQ area and CC volume during clamping are also expressed as normalized area and normalized volume, respectively. Bland–Altman analyses of the differences between normalized DEQ area and normalized CC volume of all 112 runs had an overall bias of –1.7 (5.1)% at end-systole and of 0.7 (5.0)% at end-diastole. The absolute difference was significantly greater at end-systole than at end-diastole (P<0.0001).

Figure 4 shows the same analysis for end-systole and end-diastole separately (panels A and B, respectively). Agreement was significantly less in the cava occlusion manoeuvre. The end-systolic difference was 0.5 (3.7)% and –3.9 (4.4)% during aortic and cava occlusion, respectively (P<0.0001 for absolute differences); end-diastolic difference was 1.3 (4)% and 0.2 (5.7)% during aortic and

Fig 3 Digital echo quantification (DEQ) area and conductance catheter (CC) volume measurements during interventions. (A and B) DEQ area in cm^2 (A) and CC volume in ml (B) at end of systole and end of diastole. Numerical values for the data plotted in (A) and (B) are given in Table 1. Box plots show isolated values and 10th, 25th, 50th, 75th and 90th percentiles. (C and D) Linear regression analysis of DEQ area versus CC volume at end of systole (C) and end of diastole (D). n=1680 data pairs.

cava occlusion, respectively (P<0.0001 for absolute differences).

Differences between pressure–area and pressure–volume slopes

Differences between slopes of elastance (end-systolic LV pressure in mm Hg)/(DEQ area in (cm^2×10)) and (end-systolic LV pressure in mm Hg)/(CC volume in ml) during the two types of intervention are depicted in Fig. 5. These differences were significantly but not uniformly influenced by the type of clamping (aortic or cava occlusion). Elastance slope differences were significantly smaller during aortic occlusion (mean (SD) –0.2 (3.9) than during cava occlusion

(–2.1 (8.4); P=0.03), whereas the slope differences for end-diastolic LV pressure plotted against DEQ area and CC volume, respectively, were higher during aortic occlusion (0.11 (0.85)) than during cava occlusion (0.26 (0.65); P=0.0013). The latter significance is only statistically important because the SDs of differences were very small with both manoeuvres.

Reproducibility of pressure–area versus pressure–volume slopes

For duplicate measurements, subsequent values are expressed as per cent changes from the first measurement to the second: elastance slope changes during aortic occlusion (LV

383
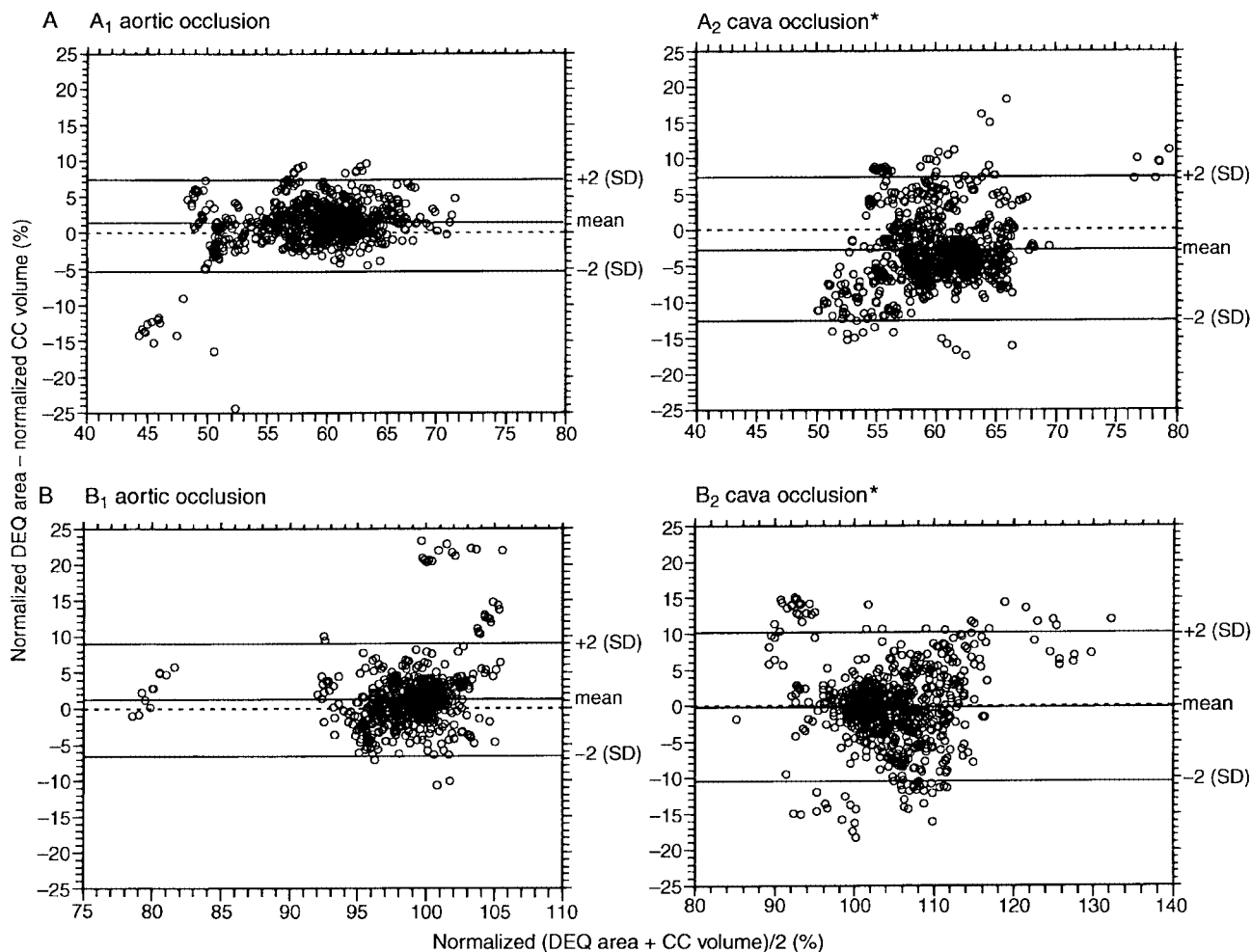
Fig 4 Bland–Altman analyses of normalized digital echo quantification (DEQ) area (%) versus normalized conductance catheter (CC) volume (%) during acute loading changes. There were 56 loading alterations with 15 data pairs each, giving 840 data pairs for aortic occlusion and 840 for cava occlusion. (A) End-systolic data pairs during aortic occlusion (A_1) and cava occlusion (A_2). (B) End-diastolic data pairs during aortic occlusion (B_1) and cava occlusion (B_2). *Absolute differences in aortic occlusion versus absolute differences in cava occlusion were significantly different ($P<0.0001$).

Table 1 Comparison of pre- and post-operative values. Data are presented as median (95% confidence interval). LV=left ventricular; DEQ=digital echo quantification; CC=conductance catheter; LVP=left ventricular pressure; FAC=fractional area shortening. *$P<0.05$ versus preoperative value. [†]$P<0.0001$ versus preoperative value.

	Preoperative		Postoperative	
	End-systolic	End-diastolic	End-systolic	End-diastolic
LVP (mm Hg)	72 (71–73)	7.5 (7.3–7.7)	73 (72–74)	8.2 (8.0–8.4)*
DEQ area (cm^2)	3.5 (3.45–3.55)	7.2 (7.1–7.3)	4.1 (4.05–4.15)[†]	7.9 (7.7–8.2)[†]
CC volume (ml)	47.7 (46.6–48.8)	125.2 (124–126.8)	59.3 (58.1–60.5)[†]	131.2 (129.0–133.4)[†]
Slope				
LVP/DEQ area ((mm Hg)/(cm^2×10))	4.1 (2.4–5.8)	0.25 (0.09–0.41)	4.5 (3.3–5.7)	0.33 (0.11–0.55)
LVP/CC volume ((mm Hg) ml^{-1})	5.4 (3.5–7.1)	0.15 (0.01–0.29)	4.7 (3.9–5.5)	0.25 (0.18–0.32)
DEQ LV FAC (%)	51.0 (49.75–50.4)	50.0 (49.8–50.5)*		
CC LV ejection fraction (%)	60.1 (59.5–60.7)	55.3 (49.7–55.9)[†]		

pressure/DEQ area 32% (95% CI 9.5–54.5%) and LV pressure/CC volume 90.6% (95% CI 25–156.2%)) were significantly greater than during cava occlusion (LV pressure/DEQ area 14.4% (95% CI –7.0 to 36.1%) ($P=0.01$) and LV pressure/CC volume 31.1% (95% CI 10.0–51.2%) ($P=0.02$)). There was no significant difference between the two clamping manoeuvres for the end-diastolic pressure–area and pressure–volume slopes: for aortic occlusion, LV
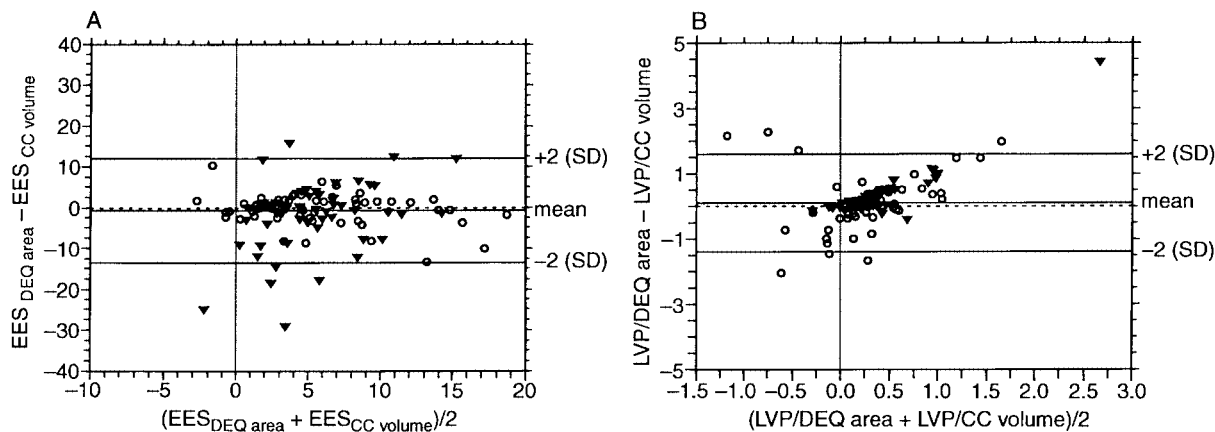
Fig 5 Bland–Altman analyses of slope differences. Differences between micromanometer left ventricular pressure (LVP)/digital echo quantification (DEQ) area ((mm Hg)/(cm^2×10)) and micromanometer LVP/conductance catheter (CC) volume ((mm Hg)/ml). Fifteen data pairs were obtained for each of 56 loading alterations, giving 840 data pairs for aortic occlusion (open circles) and 840 for cava occlusion (filled triangles). For numerical values see the text. (A) End-systolic (elastance) slope (EES) differences (*P*=0.003 aortic occlusion versus cava occlusion). (B) End-diastolic slope differences (*P*=0.0013 aortic occlusion versus cava occlusion).

pressure/DEQ area was 13.6% (95% CI –89.9 to 117.1%) and LV pressure/CC volume was 31.3% (95% CI –1.7 to 64.3%); the corresponding values for cava occlusion were 8.2% (95% CI –13.4 to 29.8%) and 28.3% (95% CI 0.3–56.3%), respectively.

Effects of CPB and atrial pacing

Differences between preoperative and postoperative results are presented in Table 1. Pooled end-systolic and end-diastolic DEQ area and CC volume values were significantly larger after CPB than before CPB, and end-diastolic LV pressure was slightly, but significantly, higher.

The end-systolic LV pressure/DEQ area and LV pressure/CC volume slopes were the same before and after CPB; the end-diastolic LV pressure/CC volume slope increase following CPB was minimal, but statistically significant. DEQ LV fractional area change, CC LV ejection fraction and thermodilution measured stroke volume all decreased significantly after CPB.

The effects of atrial pacing are summarized in Table 2. Before CPB period, a heart rate of 90 beats min^{-1}, induced by atrial pacing, led to end-systolic and end-diastolic LV pressures being lower than those at spontaneous, lower frequency. All these differences were statistically significant, but very small. After CPB, only end-diastolic LV pressure decreased significantly when atria were stimulated at 90 beats min^{-1}.

DEQ-measured LV fractional area changes with or without atrial pacing were nearly identical before and after CPB; post-CPB values differed significantly from pre-CPB values, however. CC-volume based LV ejection fraction changes were larger during atrial pacing: a 4% increase was found before CPB while a 4% reduction resulted with pacing after CPB.

End-systolic pressure–area and pressure–volume slopes were not influenced by atrial pacing, either before or after CPB. End-diastolic pressure–volume slopes decreased slightly during atrial pacing before but not after CPB.

Early postoperative outcome

No early mortality, no new regional wall motion abnormalities (as assessed by intraoperative transoesophageal echocardiography) and no perioperative myocardial infarction (as assessed by ECG and cardiac enzymes) occurred. In one patient, transoesophageal echocardiographic examination led to a non-penetrating muco-serosal lesion of the oesophagogastric transition, as diagnosed by endoscopy in a previously unknown situs inversus abdominalis. The patient was discharged from the hospital on day 11 without sequelae. One patient developed postoperative pneumonia necessitating prolonged mechanical ventilation; his hospital stay was 18 days and his condition was good at discharge. Median postoperative stay in the intensive care unit was 1.0 day (95% CI 0.1–4.4 days); median hospital stay was 9.5 days (95% CI 7.2–11.8 days).

Discussion

Consistency of methodology and findings

The DEQ system gave reliable area measurements intraoperatively in cardiac surgical patients with normal systolic LV function. During steady-state conditions, the method accurately reflects volume changes measured with the CC method; during the two types of clamping manoeuvre, end-systolic and end-diastolic area measurements remain within ±10% of the CC volume method. The area measurements

Table 2 Effects of atrial pacing. Data are presented as median (95% confidence interval). LV=left ventricular; ESP=end-systolic pressure; EDP=end-diastolic pressure; DEQ=digital echo quantification; FAC=fractional area shortening; CC=conductance catheter; EES=end-systolic elastance; ED=end-diastolic; LVP=left ventricular pressure. [†]P<0.05 sinus rhythm versus atrial pacing; [††]P<0.001 sinus rhythm versus atrial pacing; *P<0.05 preoperative versus postoperative atrial pacing; **P<0.0001 preoperative versus postoperative atrial pacing; ‡P=0.0005 preoperative versus postoperative sinus rhythm.

	Preoperative		Postoperative	
	Sinus rhythm	Atrial pacing	Sinus rhythm	Atrial pacing
LV ESP (mm Hg)	76 (74–78)	69 (67–71)[††]	74 (72–76)	71 (69–73)*
LV EDP (mm Hg)	8.1 (8.0–8.2)	7.9 (7.8–8.0)[††]	9.1 (9.0–9.2)	7.3 (7.2–7.4)[††]
DEQ LV FAC (%)	50.6 (50.1–51.4)	49.1 (48.7–49.6)[††]	51.8 (51.4–52.2)	49.0 (48.6–49.2)[††] *
CC LV ejection fraction (%)	56.8 (56–57.6)	60.9 (60.1–61.7)[††]	57.7 (56.7–58.7)	53.7 (52.9–54.5)[††] **
EES				
LVP/DEQ area ((mm Hg)/(cm^2×10))	4.1 (2.0–6.1)	4.3 (1.4–7.2)	4.4 (2.6–6.2)	4.5 (2.9–6.1)
LVP/CC volume ((mm Hg) ml^{-1})	4.5 (2.2–6.8)	7.2 (4.6–9.8)	3.8 (2.8–4.9)	5.9 (4.7–7.1)
ED				
LVP/DEQ area ((mm Hg)/(cm^2×10))	0.3 (0.05–0.55)	0.18 (0.04–0.32)	0.43 (0.03–0.83)	0.27 (0.05–0.49)
LVP/CC volume ((mm Hg) ml^{-1})	0.19 (0.01–0.37)	0.11 (–0.1 to 0.32)[†]	0.27 (0.21–0.33)	0.24 (0.13–0.33)*
Heart rate (beats min^{-1})	71 (68.5–73.5)	90 (89–91)[††]	76 (73.2–78.8)	90 (88–92)[††] ‡

obtained by the DEQ area method during cava occlusion are less precise than those obtained during aortic occlusion.

This is in accordance with the study of Chen and colleagues,[7] who used an identical echocardiography system but used a transthoracic approach for comparison with the conductance technique. In the apical four-chamber view, these authors based volume determination on automated digital echo quantification. We acquired the 2D images and, hence, the DEQ signal with a transoesophageal echo probe in a fixed position; patients were anaesthetized and measurements were taken at end-expiration. Stable images and good DEQ quality were, therefore, easier to obtain than with a hand-held transthoracic echo probe in spontaneously breathing patients. True cardiac volumes cannot be measured intraoperatively for validation of an echocardiographic volume or area determination. The CC measurements used in our study, however, have been shown to correlate with electromagnetic flow, thermodilution, contrast ventriculography, magnetic resonance imaging and ultrafast computed tomography in animals and in humans.[3 15 17] Although automatic border detection systems slightly underestimate fractional area changes by underestimating end-diastolic and overestimating end-systolic areas, compared with ultrafast computed tomography,[19] several studies have shown good correlation between the echocardiographic automated border detection and (i) real volume changes in an isolated canine heart preparation[18] and (ii) CC measurements in humans.[6 7]

Limitations of methodology and critical evaluation of results

Conductance catheter method

Theoretical and animal studies have shown that the accuracy of the CC method depends on the distance between the catheter and the wall of the ventricle, relative to the distance between the catheter injecting electrodes.[15 20–23] With larger ventricular volumes and, hence, in diastole, the method thus becomes less precise. We did not find this relevant in our study; on the contrary, differences between end-systolic volume and area measurements were significantly greater than differences at end-diastole.

Another source of error was found by Szwarc and colleagues.[27] Parallel conductance changes within cardiac cycles led to volume measurements being significantly different from those obtained by magnetic resonance imaging in an animal model. This was not considered to be of importance by Lankford and colleagues[24] or White and colleagues[25]

The metallic retractor in the open chest situation seems to be another source of artefacts for CC signals. Cabreriza and colleagues[26] found that isolation of the heart by latex wrapping could improve the reliability of volume signals. This isolation procedure, however, was introduced primarily to decrease the changes in parallel conductance, which is of importance only for absolute volume measurements. Another potential source of artefacts is the transoesophageal echo probe near the CC. We did not find that conductance signals were affected by whether the echo probe was emitting sound or not and this problem has not been reported by other investigators.

Echocardiography

During intraoperative monitoring, the mid-papillary short-axis view is widely accepted for the evaluation of LV filling state and contractility. Our aim was to validate intraoperative DEQ measurements in this standard plane against a well established method of LV volume determination. We found a small difference between the two methods within cardiac cycles. Following normalization of the DEQ area and correction for time delay, we found a cycle-specific pattern of under- and overestimation by DEQ (Fig. 3A), which supports the observations of Chen and colleagues.[7]

An important source of error in DEQ detection is the presence of the metal-containing CC in the ventricle. The echo probe had to be positioned carefully to avoid refraction and shadowing artefacts; the probe was then fixed using a specially designed apparatus. Nevertheless, DEQ area measurements were often blurred by the shadow of the CC, requiring exclusion of the catheter artefact area; particularly during the cava occlusion manoeuvre, lateral displacement of the hypovolaemic heart further impaired proper area detection. The measured LV area was therefore very small at the end of caval occlusion in a considerable number of patients, especially in those patients with small left ventricles at baseline. These artefacts probably led to spreading of the end-systolic area values during caval occlusion (Fig. 4) and the consequent spreading of elastance slope values.

Another difficulty of repeated 2D measurements is the reproducibility of the transgastric mid-papillary short-axis view. Although the probe was not moved during the measurement sequence, it cannot be guaranteed that its position was identical before and after CPB. However, each loading manoeuvre was analysed individually with regard to the area–volume relationship, and differences in area parameters before and after CPB are mirrored in CC volume measurements (Table 1).

Study patients had normal LV function without akinetic or dyskinetic wall segments and were therefore ideal for DEQ measurements. It is evident that the more regional wall motion abnormalities there are, the more bias there will be when any 2D measurements are compared with measurements of cardiac volumes.

Data analysis

Two methods were used to analyse differences between DEQ area and CC volume curves. During steady state, differences between filling and emptying patterns of LV were evaluated, the difference between each data pair being determined every 5 ms throughout the cardiac cycle. Although it would have been of interest to examine entire cardiac cycles during clamping manoeuvres too, this was not possible because of large quantities of data, so we restricted analysis of clamping manoeuvres to the clinically important time points of end-systole and end-diastole.

Calibration of the CC data for absolute volume was performed using the stroke volume derived from thermo-dilution measurements. This method is widely accepted; additional calibration with the saline dilution technique for parallel conductance did not seem to improve precision in our study. Variation of differences between normalized DEQ area and normalized CC volume data, however, was independent of calibration method.

We used a visually based, manually performed adjustment of the DEQ area curve. The time delay between registration of DEQ area and CC volume was variable and longer than reported with other automated border systems[5 9 11 25] (Fig. 1). Technically, the time delay depends on the time the system requires for calculating DEQ; physiologically, it depends on the position of the echo probe. The more apical data are acquired, the later the area curve is displayed, as compared with the CC volume curve. End-systole and end-diastole were determined manually based on the femoral arterial pressure curve (aortic valve closure) and on LV pressure. ECG was not taken into consideration as future analyses will be performed in patients with possible bundle-branch block or ventricular pacing.

Clamping manoeuvres

Partial occlusion for acute reduction in preload is widely accepted[4 5 7 9 10] and, although a partial aortic occlusion manoeuvre has been performed in animals,[22] it is not to our knowledge a standard procedure in humans. By rapidly increasing LV afterload, it assesses the capacity of the left ventricle to maintain stroke volume against an increased aortic resistance without ventricular dilation. The so-called homeometric autoregulation capability[28 29] should leave end-diastolic volume and pressure unchanged.

Reproducibility

It is difficult to repeat partial clamping of the inferior vena cava and the ascending aorta for quantitative comparison. We controlled the clamping manoeuvres on line and the surgeon tried to get a similar decrease or increase in LV pressure. Nevertheless, the resulting pressure–area and pressure–volume slopes at end-systole yielded considerable variations, those of the pressure–area loops being significantly greater.

Findings

One patient presented insufficient correlation during the caval occlusion manoeuvre despite excellent DEQ quality. One of the reasons was insufficient clamping of the vena cava inferior, which led to a relatively small decrease in LV volume, correctly measured by CC, but not detected by the DEQ method in the 2D short-axis view. The consecutive DEQ elastance revealed an opposite slope to the corresponding CC elastance slope. During aortic occlusion, only very small volume changes but important LV pressure changes are induced. Slopes are therefore determined more by the increase in LV pressure than by (the virtually unchanged) LV area and volume; the correlation of the pressure–area with the pressure–volume is therefore superior than during the cava occlusion manoeuvre.

Conclusions

We found that intraoperative changes in LV volume, assessed by CC, are reliably reflected by transoesophageal echocardiographic DEQ area changes of the LV short axis during steady-state conditions and during acute increase of afterload by partial occlusion of the ascending aorta.

When very small systolic area values resulted from partial occlusion of the vena cava, the decreased precision of the

DEQ method made it difficult to perform reproducible end-systolic LV pressure–DEQ area elastance slopes.

Patients with enlarged left ventricles and reduced ejection fraction might present more favourable conditions for LV function evaluation with vena cava occlusion manoeuvres and DEQ area measurements.

Acknowledgements

References

1 Suga H, Sagawa K. Load independence of the instantaneous pressure–volume ratio of the canine left ventricle and effects of epinephrine and heart rate on the ratio. *Circ Res* 1973; **32**: 314–22

2 Baan J, Jong TT, Kerkhof PL *et al.* Continuous stroke volume and cardiac output from intra-ventricular dimensions obtained with impedance catheter. *Cardiovasc Res* 1981; **15**: 328–34

3 Baan J, van der Velde ET, de Bruin HG *et al.* Continuous measurement of left ventricular volume in animals and humans by conductance catheter. *Circulation* 1984; **70**: 812–23

4 Schreuder JJ, Biervliet JD, van der Velde ET *et al.* Systolic and diastolic pressure–volume relationships during cardiac surgery. *J Cardiothorac Vasc Anesth* 1991; **5**: 539–45

5 Gorcsan J III, Denault A, Mandarino WA, Pinsky MR. Left ventricular pressure–volume relations with transesophageal echocardiographic automated border detection: comparison with conductance-catheter technique. *Am Heart J* 1996; **131**: 544–52

6 Nomura K, Kurosawa H, Morita K *et al.* Comparison of left ventricular pressure–volume loops measured by two-dimensional echocardiography and a conductance catheter. *Nippon Kyobu Geka Gakkai Zasshi* 1997; **45**: 37–41 (English abstract)

7 Chen CH, Nevo E, Fetics B *et al.* Comparison of continuous left ventricular volumes by transthoracic two-dimensional digital echo quantification with simultaneous conductance catheter measurements in patients with cardiac diseases. *Am J Cardiol* 1997; **80**: 756–61

8 Deneault LG, Kancel MJ, Denault A *et al.* A system for the on-line acquisition, visualization, and analysis of pressure–area loops. *Comput Biomed Res* 1994; **27**: 61–7

9 Gorcsan J III, Romand JA, Mandarino WA, Deneault LG, Pinsky MR. Assessment of left ventricular performance by on-line pressure-area relations using echocardiographic automated border detection. *J Am Coll Cardiol* 1994; **23**: 242–52

10 Gorcsan J III, Gasior TA, Mandarino WA, Deneault LG, Hattler BG, Pinsky MR. Assessment of the immediate effects of cardiopulmonary bypass on left ventricular performance by on-line pressure-area relations. *Circulation* 1994; **89**: 180–90

11 Gorcsan J III, Denault A, Gasior TA *et al.* Rapid estimation of left ventricular contractility from end-systolic relations by echocardiographic automated border detection and femoral arterial pressure. *Anesthesiology* 1994; **81**: 553–62

12 Denault AY, Gorcsan J III, Mandarino WA, Kancel MJ, Pinsky MR. Left ventricular performance assessed by echocardiographic automated border detection and arterial pressure. *Am J Physiol* 1997; **272**: H138–47

13 Mandarino WA, Pinsky MR, Gorcsan J III. Assessment of left ventricular contractile state by preload-adjusted maximal power using echocardiographic automated border detection. *J Am Coll Cardiol* 1998; **31**: 861–8

14 Bednarz JE, Marcus RH, Lang RM. Technical guidelines for performing automated border detection studies. *J Am Soc Echocardiogr* 1995; **8**: 293–305

15 Szwarc RS, Laurent D, Allegrini PR, Ball HA. Conductance catheter measurement of left ventricular volume: evidence for nonlinearity within cardiac cycle. *Am J Physiol* 1995; **268**: H1490–8.

16 Odake M, Takeuchi M, Takaoka H, Hata K, Hayashi Y, Yokoyama M. Determination of left ventricular volume using a conductance catheter in the diseased human heart. *Eur Heart J* 1992; **13** (Suppl E): 22–7

17 Akaishi M, Matsubara T, Abe S, Goto S, Yokozuka H, Handa SC. Conductance catheter for determining left ventricular volume and end-systolic pressure–volume relations: its clinical application. *J Cardiol* 1992; **22**: 539–47

18 Gorcsan J III, Morita S, Mandarino WA *et al.* Two-dimensional echocardiographic automated border detection accurately reflects changes in left ventricular volume. *J Am Soc Echocardiogr* 1993; **6**: 482–8

19 Marcus RH, Bednarz J, Coulden R, Shroff S, Lipton M, Lang RM. Ultrasonic backscatter system for automated on-line endocardial boundary detection: evaluation by ultra fast computed tomography. *J Am Coll Cardiol* 1993; **3**: 839–47

20 Woodard JC, Bertram CD. Effect of radial position on volume measurements using the conductance catheter. *Med Biol Eng Comput* 1989; **27**: 25–32

21 Steendijk P, Van der Velde ET, Baan J. Left ventricular stroke volume by single and dual excitation of conductance catheter in dogs. *Am J Physiol* 1993; **264**: H2198–207

22 Boltwood CM, Appleyard RF, Glantz SA. Left ventricular volume measurement by conductance catheter in intact dogs. *Circulation* 1989; **80**: 1360–77.

23 Hettrick DA, Battocletti JH, Ackmann JJ, Linehan, Warltier DC. Effects of physical parameters on the cylindrical model for volume measurements by conductance. *Ann Biomed Eng* 1997; **25**: 126–34

24 Lankford EB, Kass DA, Maughan WL, Shoukas AA. Does volume catheter parallel conductance vary during a cardiac cycle? *Am J Physiol* 1990; **258**: H1933–42

25 White PA, Chaturvedi RR, Shore D, Lincoln C, Szwarc RS, Bishop AJ *et al.* Left ventricular parallel conductance during cardiac cycle in children with congenital heart disease. *Am J Physiol* 1997; **273**: H295–302

26 Cabreriza SE, Dean DA, Jia CX, Dickstein ML, Spotnitz HM. Electrical isolation of the heart. Stabilizing parallel conductance for left ventricular volume measurement. *ASAIO J* 1997; **43**: M509–14

27 Szwarc RS, Mickleborough LL, Mizuno SI, Wilson GJ, Liu P, Mohamed S. Conductance catheter measurements of left ventricular volume in the intact dog: parallel conductance is independent of left ventricular size. *Cardiovasc Res* 1994; **28**: 252–8

28 Sarnoff SJ, Mitchell JH, Gilmore JP, Remensnyder JP. Homeometric autoregulation in the heart. *Circ Res* 1960; **8**: 1077–91

29 Baan J, Van Der Velde ET, Steendijk P. Ventricular pressure–volume relations *in vivo*. *Eur Heart J* 1992; **13** (Suppl E): 2–6.